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QUANTITATIVE APEXCARDIOGRAPHIC INDEX "(PEAK DA/DT)/A"

— Experimental and clinical studies —

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Abstract

We evaluated the efficacy and the limitation of the apexcardiographic index "(peak dA/dt)A" by animal and clinical observations and obtained the following results.

1. Since significant correlations were obtained between (peak dA/dt/A and Vmax (r=+0.83), dp/dt/p (p=50mmHg) (r=+0,85), it was suggested that this parameter might be useful for the assessment of left ventricular contractility.

2. While this parameter appeared to be affected by afterload, it would be hardly affected by preload.

3. The normal value was 23 ± 4 sec^{-1}. It was not influenced by heart rate, but by the posture at recording of the apexcardiogram.

4. In clinical observations, it was also postulated that this parameter might be an useful index to evaluate cardiac status and severity.

INTRODUCTION

Apexcardiography has been used as one of the noninvasive methods to evaluate simply the left ventricular function. Since the ascending portion from the C point of the apexcardigram (ACG) coincides especially well with that of the left ventricular pressure curve, attemts are being made to evaluate the left ventricular function from the first derivative of ACG (ΔACG)[1-7]. In view of our deep interest in the quantitative apexcardiographic index—(peak dA/dt)/A—proposed by Motomura et al[6]., we studied the efficacy and the limitation of their method by animal and clinical observations.

SUBJECTS AND METHOD

1) Animal experiment

Using 15 mongrel dogs (10-25 kg), anesthetized with pentobarbital (30 mg/kg), the following procedures were introduced. A catheter-tipped

micromanometer was inserted into the left ventricle from the right carotid artery and into the aortic arch from the femoral artery both in the retrograde fashion. The left ventricular pressure, its first derivative (dp/dt), and the aortic pressure were simultaneously recorded with ACG and ⊿ACG in the left lateral position. These tracings were made at the paper speed of 200 mm/sec.

After the above preparatory interventions isoproterenol (20-50 μg) or propranolol (10 mg) was administered intravenously in order to alter the cardiac contractility. The mutual relationship between the change in (peak dA/dt)/A and each invasively derived parameter was studied. Of the parameters obtained invasively, both Vmax (by Mason's method)[8], and dp/dt/p were measured by the developed pressure. The administration of these drugs was done on 10 dogs at intervals of 20 to 30 minutes.

In order to study the effects of preload and afterload, intravenous injection of 500 ml dextran (10 animals) and of 1-2 mg methoxamine (7 animals) were given. In this instance, the heart rate was kept constant by the right atrial pacing.

2) Clinical observation

The control group was consisted of 30 normal persons (age ranging 21-60 years), 31 patients with ischemic heart disease (IHD) (44-80 years) and with myocardial disease (MD) (21-68 years), 34 with hypertension (HT) (25-74 years), 3 with congenital heart disease (CHD) (24-40 years), 12 with valvular heart disease (VHD) (24-74 years) and 7 with hyperthyroidism, to the total of 117 as the subjects.

After 15-minute bed rest, ACG and ⊿ACG were recorded at the paper speed of 100 mm/sec in midexpiratory apnea and in the left lateral position at about 60°.

Recording of ACG were taken with transducer of TY 303 (Fukuda Electronic Co.) and of ⊿ACG with RC circuit at the time constant of 1 msec. for both basic and clinical studies. The measurement of (peak dA/dt)/A was made by the method of Motomura et al. as shown in Fig. 1 for simultaneous recording of ACG and ⊿ACG. Peak dA/dt was divided by A that is the height of ACG at the same time from the C on ACG. All the measurements were represented by the average values of 5 heart beats.

RESULTS

1. Animal experiments

1) Change in the contractility

Observations were carried out on the changes of (peak dA/dt)/A at

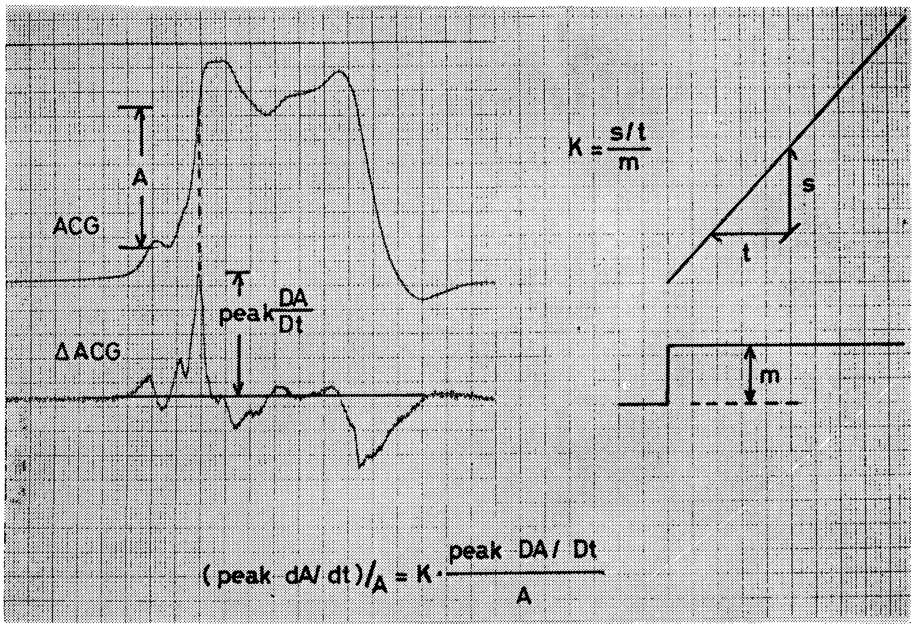

Fig. 1 How to measure (peak dA/dt)/A

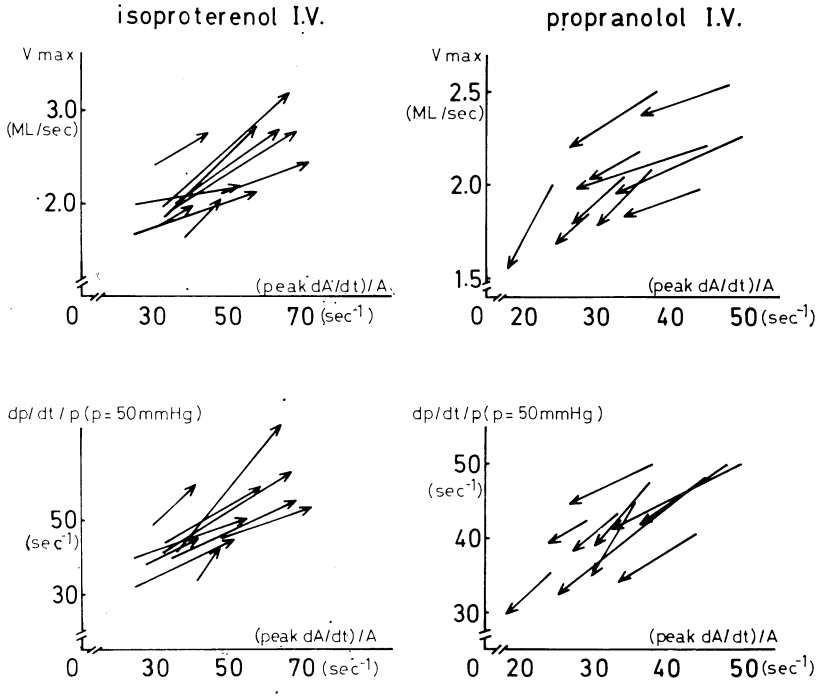

Fig. 2. Relationship of changes in (peak dA/dt)/A to Vmax and to dP/dt/p at the time when isoproterenol or propranolol was administered.

the time when hemodynamics were altered by the administration of isoproternol and propranolol, and the mutual relationships between (peak dA/dt)A and the several invasive parameters (Vmax, dp/dt/p, max dp/dt, left ventricular end-diastolic pressure-LVEDP) were assessed.

Fig. 2 shows relationships of changes in (peak dA/dt)/A to Vmax and to dp/dt/p at the time when the above two drugs were administered. The diagrammatic representation indicates that the parameter after isoproterenol administration was increased in the same direction as Vmax and as dp/dt/p. On the other hand, the parameter was decreased in the same direction as Vmax and as dp/dt/p after propranolol.

Fig. 3 illustrates the relationship of (peak dA/dt)/A to Vmax, dp/dt/p, max dp/dt, and LVEDP when the contractility was altered.

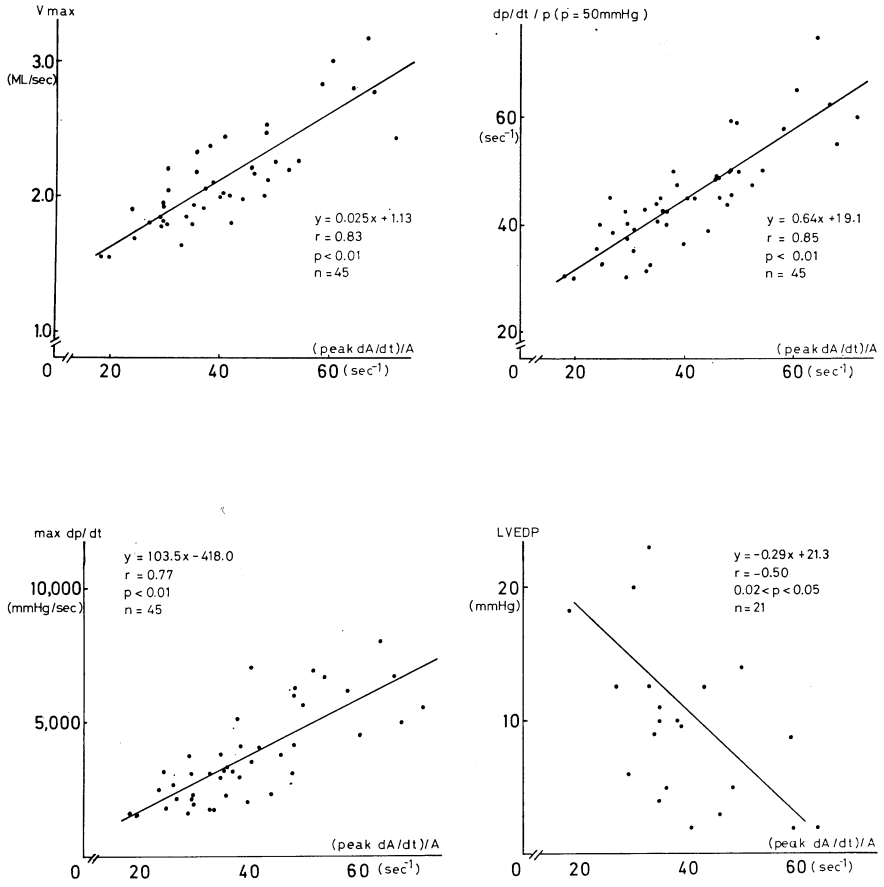

Fig. 3. Relationship of (peak dA/dt)/A to various invasive parameters when the contractility was altered.

The correlation coefficient to Vmax was +0.83, to dp/dt/p was +0.85, to max dp/dt was +0.77 and to LVEDP was −0.50, respectively.

2) Changes of afterload

After intravenous administration of 1 to 2 mg of methoxamine, the change of (peak dA/dt)/A was observed (Fig. 4-A). When the aortic diastolic pressure rose by 17% (21mmHg) in average, the parameter was increased by 11% ($p < 0.05$) in average, and when it rose by 39% (35 mmHg) in average, the parameter was increased by 16% ($p < 0.05$).

3) Changes of preload

After rapid intravenous adminitrations of 500ml of dextran, changes of (peak dA/dt)/A were studied (Fig. 4-B). It was found that when

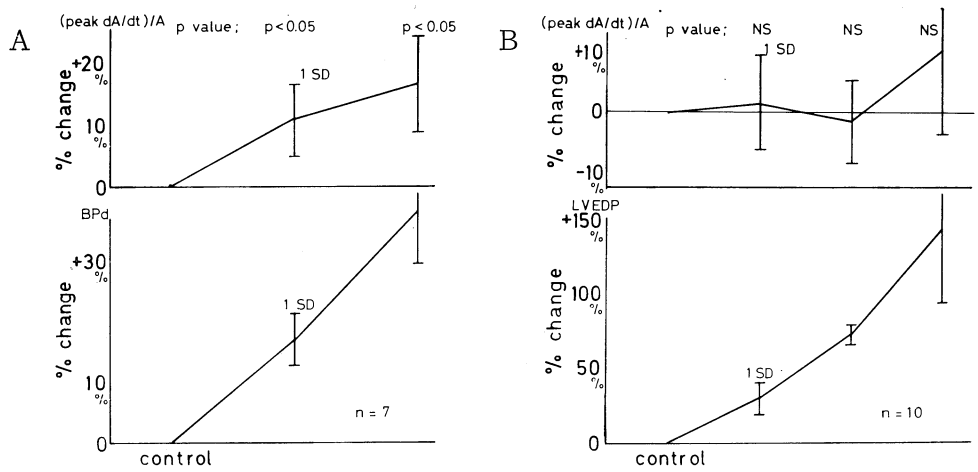

Fig. 4. Effect of afterload and preload on (peak dA/dt)/A.
A: alteration in afterload (by methoxamine 1-2 mg I. V.)
B: alteration in preload (by dextran 500 ml I. V.)

LVEDP rose by 14.2% (17mmHg) this parameter tended to rise but its elevation was not so significant. After the dextran administration the aortic diastolic pressure was decreased in the whole, but its fall was 5 mmHg in average.

2. Clinical study

Fig. 5 gives the relationship between the heart rate and (peak dA/dt)/A in 30 normal individuals. It was found this parameter not to be affected by the heart rate. The normal value (mean$\pm$2 SD) was 23 ± 4^{-1}. For the purpose to determine the effect of the patients posture on (peak dA/dt)/A, tracings were made from the same patient in various postures

such as supine and left lateral at the angles of 30°, 45°, 60°, and 80°. Fig. 6 gives the values at various postures expressed in per cent representing the posture inclied 60° to the left as 0%. The measured values gave the lowest at the supine position and as the angle to the left lateral position increased, its value was augmented. Taking to 60° to

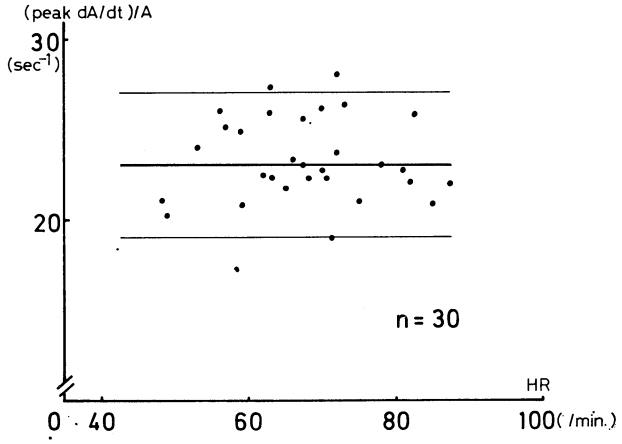

Fig. 5. Relationship between the heart rate and (peak dA/dt)/A in normal individuals.
Bars indicate mean value±2 standard deviations.

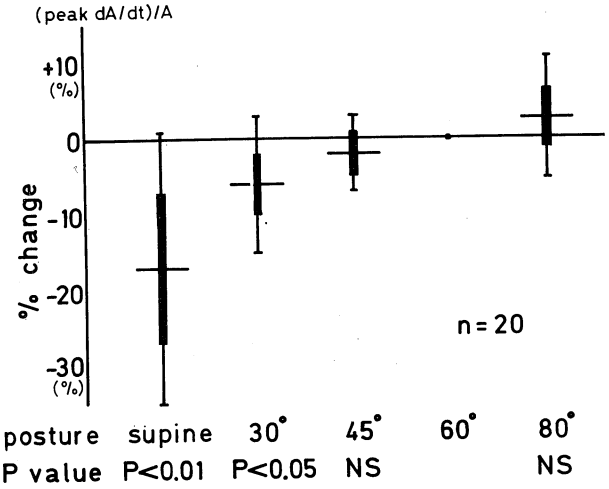

Fig. 6. Changes of (peak dA/dt)/A in various postures.
Bars indicate mean value±1 standard deviation.
Thick columns indicate 95% confidence limit.
P values: between 60° and each posture.

the left as standard, at the 45° the value tended to be decreased and at the 80° it tended to be increased, but in either case there could be recognized no significant difference. However, this value at the supine position and at the 30° to the left, there could be recognized a significant decrease.

Fig. 7 is the diagrammatic representation of individual values, their average values, and 95% confidence limits. The average value of the normal group was 23 sec^{-1}, and that of IHD and MD groups was 18.2, showing a significant decrease. In addition, in CHD and VHD groups it was 18.6, indicating a significant decrease, while in hyperthyroidism it was 28.6, showing a significantly high value. The HT group tended to give a high value of 25.3.

Fig. 8 shows the results of various heart disease groups (except HT and hyperthyroidism) classified according to the severity of New York Heart Association. As the severity advanced, the average value tended to decline significant.

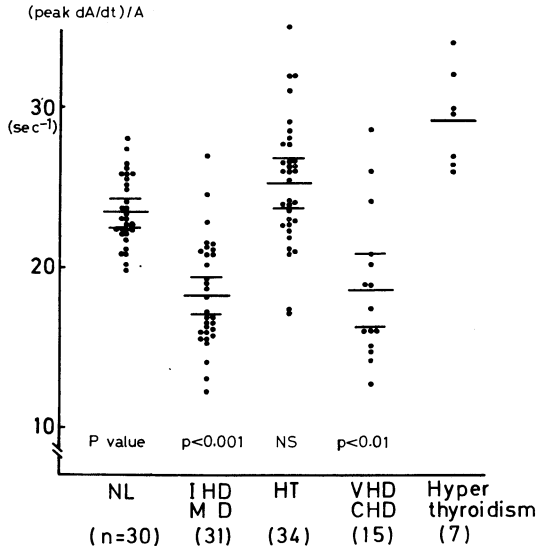

Fig. 7. (peak dA/dt)A in various heart disease.
Bars indicate mean value ±95% confidence limit.
NL=normal, IHD=ischemic heart disease
MD=myocardial disease, HT=hypertension
VHD=valvular heart disease. CHD=congenital heart disease
P value=between normal group and each heart disease group

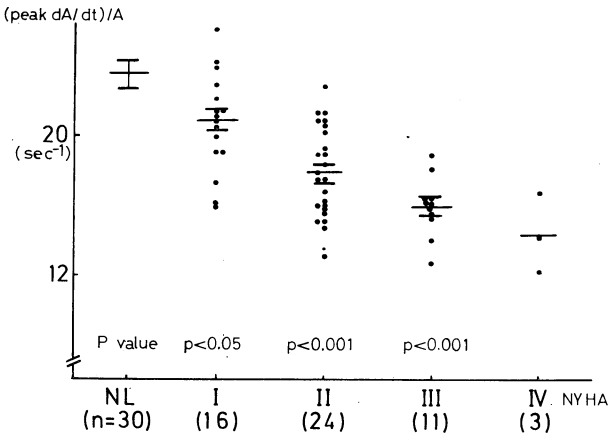

Fig. 8. (peak dA/dt)/A in various cardiac functional classes (NYHA classification)
P value=between normal group and each cardiac class group

DISCUSSION

Recently the first derivative of apexcardiogram (*Δ*ACG) has been presented as one of the useful noninvasive methods in evaluating the left ventricular function. Johnston et al.[8] stated as early as in 1951 that using the amplitude of the *Δ*ACG the left ventricular function was able to assess in its relation to the motion velocity of the left ventricular wall. However, since there is a great individual difference in the size of the heart and in the coupling with thoracic wall, the problem of quantification could not be solved so that their method has not been generally applied in general practice. Willems et al.[3] recognized a good correlation between the first derivative of left ventricular pressure (dp/dt) and *Δ*ACG in animal experiments. On the other hand, Reale[1] measured the time between R wave on ECG and peak *Δ*ACG (t-ACG), and stated that this method enabled to evaluate quantitatively the left ventricular function and it did not require calibration of ACG. Vetter[2] following Reale's idea, also measured the time from the beginning of Q wave on ECG to the peak of *Δ*ACG and obtain the similar results. Denet et al.[5] conducted the quantification of ACG using the water-filled chamber in animal experiment and found that *Δ*ACG reflected well the contractility of the heart muscle. Motomura[6] and Mirsky[4] reported to estimate quantitatively the amplitude of the ACG using the relative values of amplitudes of ACG.

We evaluated the method by Motomura et al., which was throught

to be relatively simple, in order to determine its efficacy and its limitation. They have studied the correlation between this parameter and the left ventricular function as obtained invasively with thirty-two patients. Whereas in our study we studied the correlation of this parameter and the one obtained invasively after altering hemodynamics with animals and also with many clinical cases.

In the animal experiments isoproterenol administration raised (peak dA/dt)/A, while propranolol decreased this parameter. In addition, (peak dA/dt)/A revealed strong positive correlation with both Vmax ($r=+0.83$) and dp/dt/p ($r=+0.85$) that has been reported to represent the cardiac contractility. From these findings it may be suggested that this parameter could be useful as an index which reflects the contractility.

For the purpose to study effects of preload and afterload 500 ml of dextran and to 2 mg of methoxamine were administered. When LVEDP rose by 14.2% (17 mmHg) in average after dextran, this parameter tended to rise but not significantly, and the fall of aortic diastolic pressure was also slight, suggesting that this parameter may not be affected by preload.

In contrast, with the administration of methoxamine the aortic diastolic pressure rose by 21 mmHg in average while this parameter fell 11% in average which was significant, and on the average rise by 35 mmHg of diastolic pressure, the parameter fell 16% in average which was also significant. Consequently, this parameter seems to be affected by the afterload.

As to the clinical observations, shown in Fig. 5, the normal value of (peak dA/dt)/A was 23 ± 4 sec^{-1}, and it was demonstrated that this parameter was not affected by the heart rate.

In studying changes of (peak dA/dt)/A according to the posture as the subjects in supine to the left lateral position, this parameter was increased. Therefore, this parameter might be affected by the posture. It is reported[10-12] that the ACG was changed by the posture, and in the left lateral position A-wave and systolic wave form were increased. This seems to be due to the fact that the coupling of the thoracic wall with the heart is changed by the body position. In any case the change of the ACG pattern caused by the posture cannot be avoided, so that in order to eliminate such a change, it is necessary to keep the posture of the subject approximately constant.

In the groups of IHD, MD, CHD and VHD the (peak dA/dt)/A showed a significant decrease as comparaed with the normal group. It

is reported[13-15] that in these heart disease the contractility was decreased as a group, and our results would also coincide with these findings. On the other hand, the fact that this parameter was increased in hyperthyroidism might be related to the acceleration of the cardiac contractility[16-17]. As mentioned above, the (peak dA/dt)/A stoichiometrically distinguished among the normal group on one hand and heart disease groups on the other.

In the HT group this parameter tended to be increased, though not significant, as compared with the normal group. In the HT the left ventricle may be hypertrophied, resulting in the fall of the contractility[18,19]. Furthermore, the sympathetic tone might be accelerated in HT so that there must be caused with acceleration of the contractility[20,21], hence the evaluation of the left ventricular function might be complex. In addition to it this parameter seems not to eradicate the effect of afterload as demonstrated in animal experiments. From these points the data that we have obtained in the present study concerning HT are difficult of interpretation and further studies would be required.

Furthermone as shown in Fig. 8, this parameter was decreased according to the severity of the heart disease classified by New York Heart Association so that the severity of heart disease may be estimated to a certain extent. Nonetheless, there could be observed a considerable number of values that overlaped between the normal group on one hand and NYHA 1 or II (of less severe groups) on the other, so that to distinguish patients with mild heart disease from normal subjects it will require some kind of provocative testing. This measurement has a limitation in that it may not be applied to patients with pulmonary emphysema or obesity. With most of cases with heart disease, however, the recording of the ACG would be possible, hence the percentage of successful measurement should be considered to be high.

CONCLUSION

Concerning the (peak dA/dt)/A we have conducted animal and clinical observations and obtained the following results.

1. It was demonstrated that this parameter would be representing noninvasively the contractility of the heart.

2. While it was not possible to eradicate effects of the afterload it was considered not possibly affected by preload.

3. The normal value was 23 ± 4 sec^{-1} and this parameter was not influenced by the heart rate. However, the recordings should be taken at a constant posture.

4. Since this parameter enabled us to differentiate stoichiometrically normal group from groups of various heart disease, and its findings coincided also with the severity of the disease, it may serve as one of clinical parameters.

5. As for the group of hypertension further studies seem to be required.

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